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Evolution of edema, hemorrhage and microvascular obstruction after acute myocardial infarction

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Introduction

In acute myocardial infarction (AMI), the no-reflow phenomenon is caused by ischemia-induced microvascular injury/obstruction and has been correlated with adverse remodeling. The severity of the initial ischemic insult may also lead to intramyocardial hemorrhage. Alongside, intracellular and interstitial edema is a consistent feature of AMI and has been associated with the salvageable area-at-risk. The (in-vivo) evolution of these processes throughout infarct healing is not well-characterized but is important in grading severity and evaluating treatment strategies, potentially improving clinical outcome.

Purpose

To characterize the time course of edema (T2), hemorrhage (T2*) and microvascular obstruction (MVO) in porcine myocardium following AMI and observe the relative resolution of these pathophysiological mechanisms.

Methods

7 pigs underwent MRI before LAD infarction (control) with subgroups studied at 2,7,14, and 30-42 days post-infarction. Histology was performed upon sacrifice at either Day 14 (n = 3) or Day 30-42 (n = 4). Imaging was performed on a 3 T MRI scanner (MR 750, GE Healthcare). A previously validated T2-prepared spiral sequence was utilized for T2 quantification and T2* was determined using a multi-echo gradient-echo acquisition. An early (~3 min) contrast-enhanced (CE) IR-GRE sequence was used for infarct/MVO delineation. Diastolic-wall-thickness (DWT) was measured from CINE-SSFP imaging.

Results

Figure 1 demonstrates T2, T2* maps and early CE images for an anterio-septal infarct in a short-axis slice for a representative animal at three time points. T2-maps represent edematous changes (bright regions), T2*-maps indicate hemorrhage (dark regions) while CE images delineate MVO (signal voids within infarct). Figure 2 shows the cumulative time course of T2, T2* and DWT within the infarct. T2 was indistinguishable from control at day 2 ($p = 0.38$) while the T2 elevation beyond week 1 was statistically significant ($p < 0.05$). T2* was reduced up to week 1 as a result of hemorrhage and its normalization at week 4 coincided with resolution of MVO. DWT was significantly increased at day 2 (7.5 vs 5.3 mm, $p = 0.06$) suggesting increased tissue water content while it fell below control values at week 6 (4.3 mm, $p = 0.003$) indicating scar formation.

Conclusion

Post-infarct remodeling is a complex process and comparison with remote myocardium is equally important. In this respect quantitative T2 and T2* mapping techniques are potentially more specific than intensity measures in single images. Edema and hemorrhage have counter-acting effects on T2, hence care should be taken while evaluating day 2. Our study demonstrates that multi-factorial

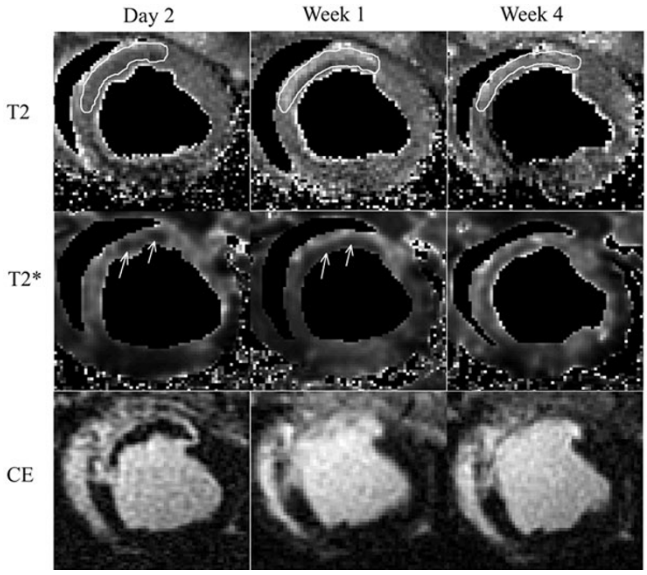

Figure 1
At day 2 in this animal, T2 elevation usually associated with edema was not apparent in the infarct zone (39.2 ms vs 39.1 ms contrl); however DWT was increased by 34% suggesting edematous swelling. Lower T2* (arrows) indicated presence of hemorrhage (18.5 ms vs 34.2 ms whle the CE image showed a large MVO. At Week 1, T2 was elevated in most of the infarct (51.1 ms) with reduced T2* (20.5 ms) indicating diffuse hemorrhagic by-products. CE imge showed only a slight MVO. By week 4, hemorrhage/MVO were resolved.

MR-based parameters, acquired in a longitudinal fashion, can be employed to assess the evolution of myocardial infarction.

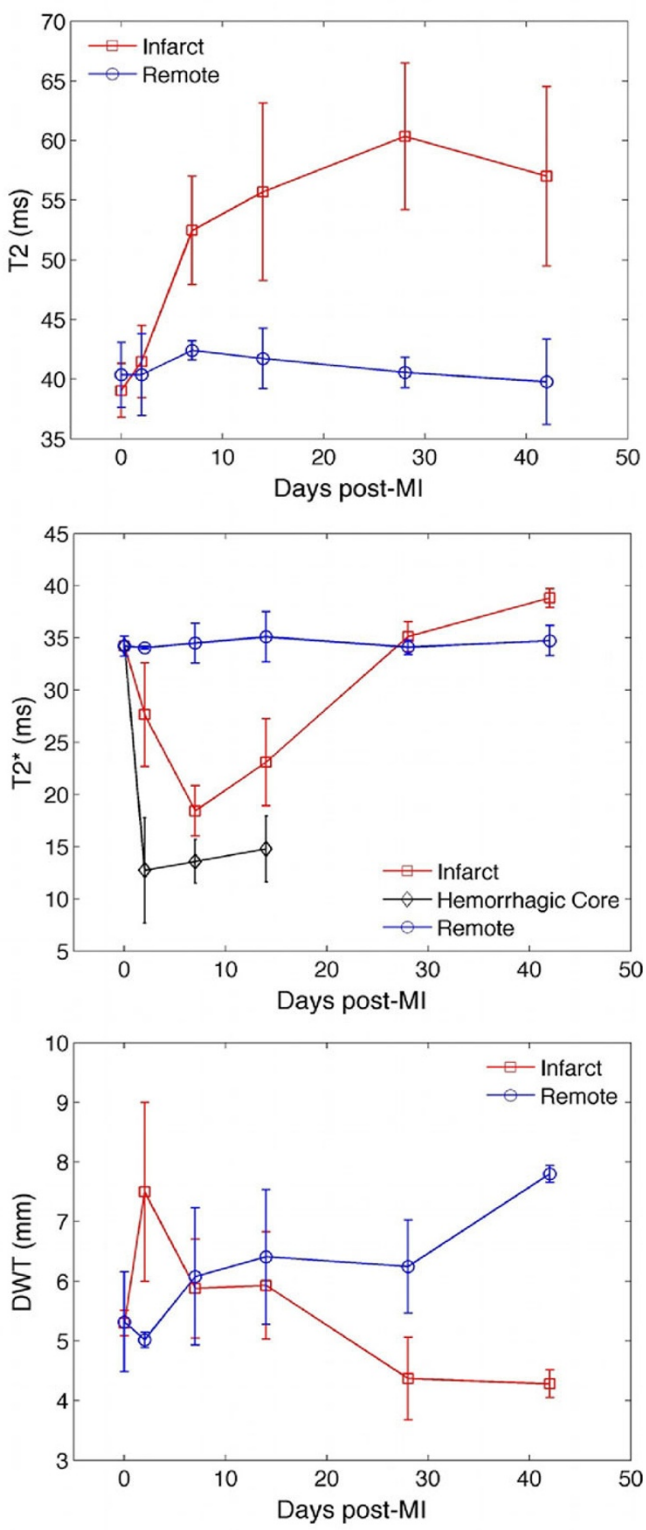

Figure 2
Plots demostrate longitudinal fluctuations in T2, T2* and DWT in infarct zone compared to remote myocardium averaged over all animals